

Migraine

The danger of ignoring a migraine

N P S Bajaj, P K Morrish

Answers on p 57.

A 31 year old man was referred with prolonged loss of vision after a typical migraine attack. His usual migraine attacks consisted of a left sided headache preceded by nausea and vomiting and a visual aura of coloured and flashing lights in both visual fields. Visual loss after his migraines could last up to one hour.

The frequency of his headaches had markedly increased eight months before presentation and these were relieved with a combination of pizotifen and naratriptan. Three weeks before presentation, he suffered a typical migraine but noticed that the visual loss after the migraine persisted as a "hole" in the vision of the left eye. Two weeks later he developed a similar scotoma in the field of the right eye with distortion of vision in that eye such that straight lines

appeared wavy. He had no other relevant past medical history.

On examination, his visual acuity was restricted to perception of movement only in the left eye and 6/60 in the right eye. Fundoscopy revealed the presence of arteriolar-venous nipping, "flame shaped" and "dot and blot" retinal haemorrhages, "cotton wool" spots, and papilloedema (see fig 1C and 1D). Visual field assessment revealed the presence of bilateral scotomata. The remainder of his clinical examination was unremarkable, although his blood pressure was found to be markedly raised at 220/180 mm Hg.

Blood tests including electrolytes and full blood count were normal. Computed tomography of the brain showed symmetrical hypodense areas in the posterior temporal lobes. These hypodense areas seen on computed tomography

appeared as increased signal on T2 weighted magnetic resonance imaging (MRI); further areas of increased T2 signal, not seen on the computed tomogram, were revealed in both hemispheres (fig 1A and 1B). Repeat MRI of the brain one month later was normal.

QUESTIONS

- (1) What is the term used to describe the MRI brain appearance in fig 1?
- (2) Give the underlying cause of the brain appearance in this case.
- (3) Give three other causes of this condition.
- (4) What is the likely cause of the fundal appearance in fig 1?
- (5) What other investigations would you order?

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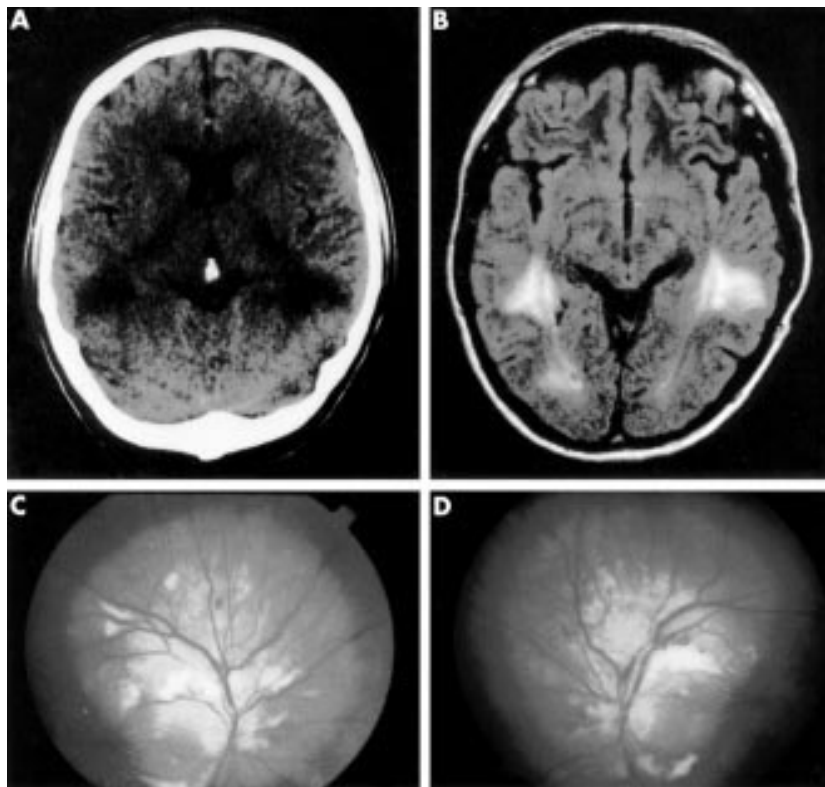


Figure 1 (A) Computed tomography of the brain and (B) FLAIR (fluid attenuated inversion recovery) sequence MRI brain shows symmetrical change in the posterior parietal and occipital lobes (seen as decreased signal on computed tomography of the brain and increased signal on FLAIR MRI). (C) and (D) illustrating fundoscopic changes including "cotton wool spots", hard exudates, arteriolar-venous nipping, "flame shaped" and "dot and blot" haemorrhages and papilloedema.

Hyperkalaemia

Hyperkalaemia in an elderly diabetic patient

J V Butler, M Hasan

Answers on p 57.

A 76 year old man was referred to casualty with a non-haemolysed serum potassium of 6.6 mmol/l. He was entirely asymptomatic. He had a past medical history of type II diabetes mellitus with diabetic nephropathy, hypercholesterolaemia, and cerebrovascular disease with a residual left hemiparesis from a previous stroke. He had recently increased his coffee intake and had started drinking four glasses of orange juice daily. His medications were aspirin 75 mg daily, cerivastatin 100 µg daily, gliclazide 160 mg twice a day, and nebivolol 5 mg daily (recently started for newly diagnosed hypertension).

On examination his blood pressure was 172/88 mm Hg and he had both left upper and lower limb weakness consistent with a previous stroke. He had some evidence of peripheral neuropathy but nothing to suggest a proximal myopathy. Examination was otherwise unremarkable. His electrocardiograph showed peaked T waves in most leads.

Initial investigations showed: serum sodium concentration 145 mmol/l, serum potassium 6.6 mmol/l, urea 11.1 mmol/l, creatinine 172 µmol/l, glucose 4.5 mmol/l, albumin 36 g/l, glycated haemoglobin (HbA_{1c}) 8.3% (reference range 4.5–6.5). A 24 hour urinary creati-

nine clearance was 56 ml/min while 24 hour urinary protein was raised at 1.67 g/24 hours. Serum aldosterone was 100 (100–450) pmol/l and serum renin was <0.2 (1.1–2.7) pmol/ml/hour. One year previously his biochemistry showed: serum sodium 143 mmol/l, potassium 5.4 mmol/l, urea 9.9 mmol/l, creatinine 143 µmol/l, and glucose 5.9 mmol/l.

QUESTIONS

- (1) What are the factors causing his hyperkalaemia?
- (2) Why are elderly patients prone to developing hyperkalaemia?
- (3) How would you manage this patient?

Postgrad Med J 2002;78:54

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Occlusive vasculitis

Recurrent orogenital ulcers with papilloedema and headaches

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Answers on p 57.

A 33 year old man with a past history of recurrent orogenital ulcers, generalised arthritis, eye inflammation, and an episode of deep vein thrombosis presented with a three day history of worsening headaches not responding to simple analgesia.

On examination he was pyrexial with a temperature of 38°C. Neurological examination showed no signs of meningism or any focal neurological deficit, but fundus examination showed papilloedema. Other systemic examinations were unremarkable.

Initial investigations including full blood count, urea and electrolytes, chest radiography, and urinalysis were normal. Erythrocyte sedimentation rate was 15 mm/hour and C reactive protein was less than 7 mg/l. In view of the fundal findings and headaches, the patient had unenhanced computed tomography of his brain (see fig 1).

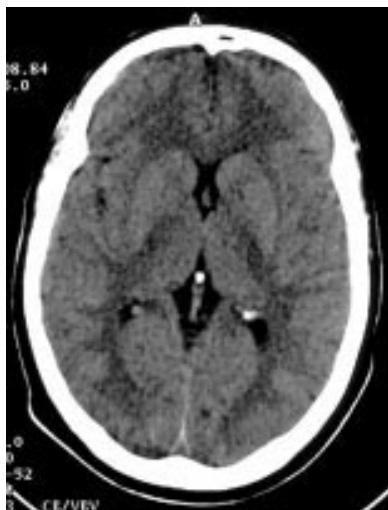


Figure 1 Unenhanced computed tomogram of brain.

QUESTIONS

- (1) What is the initial diagnosis?
- (2) What does the unenhanced computed tomogram of the brain show?
- (3) What probable complication of the initial diagnosis has occurred?
- (4) What further investigation will you do to confirm the complication?
- (5) What is the treatment of this complication?

Postgrad Med J 2002;78:54

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Pulmonary disease

Malaise, weight loss, and respiratory symptoms

H A Hadi, A G Arnold

Answers on p 58.

A 58 year schoolteacher was admitted with a two month history of malaise, weight loss, worsening dyspnoea, and dry cough. There was no improvement after a course of antibiotics. There were no symptoms of arthralgia, wheezing, or fever. The patient, a non-smoker, had no history of exposure to animals and no relevant occupational or family history. She was generally in good health, apart from recurrent urinary tract infections.

On clinical examination she was apyrexial, with a respiratory rate of 20 breaths/min and an oxygen saturation of 90% on air. Examination of the cardiovascular, abdominal, and musculoskeletal systems was unremarkable. Chest examination showed decreased expansion and bilateral basal inspiratory crackles on auscultation.

Serum electrolytes, renal function, and urinalysis results were normal. Blood culture was negative. A blood count showed a haemoglobin concentration of 140 g/l and a white cell count of $12.5 \times 10^9/l$ (neutrophils 84%, eosinophils 0.6%). The erythrocyte sedimentation rate (ESR) was 51 mm/hour. The electrocardiogram was unremarkable, and arterial blood gas measurements while breathing room air were normal. Lung function tests showed a forced expiratory volume in one second of 1.52 litres (55% of predicted) and vital capacity of 1.76 litre (54% of predicted). Flow volume loops were abnormal (fig 1). Imaging investigation included a chest radiograph (fig 2) and a high resolution computed tomography (HRCT) scan of the thorax (fig 3).

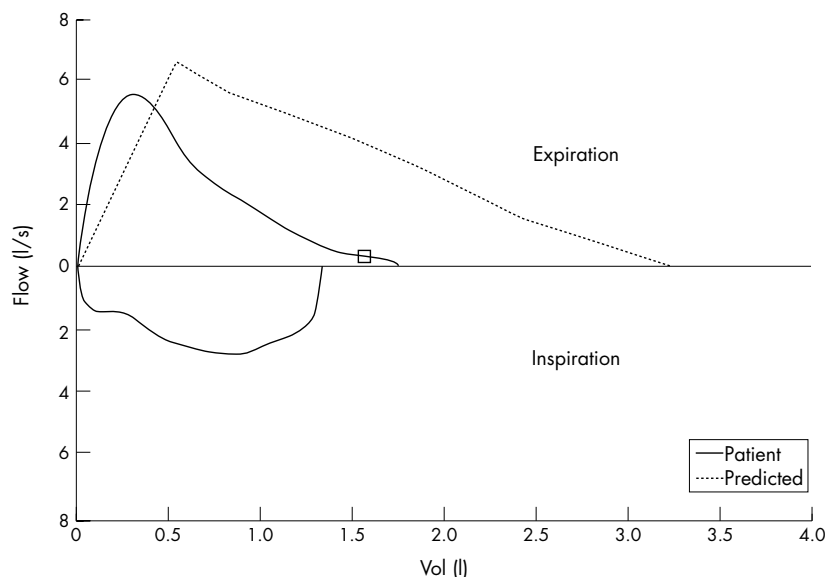


Figure 1 Flow volume loop.

QUESTIONS

- (1) What abnormalities are shown in the flow volume loops?
- (2) What abnormalities are shown in the chest radiograph and computed tomogram?
- (3) What is the differential diagnosis?

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Figure 2 Chest radiograph.

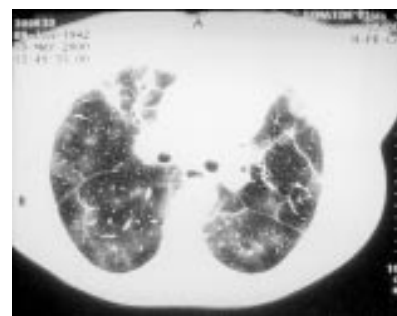


Figure 3 HRCT scan of the thorax.

Jaundice

Jaundice in primary school pupils

I Stephenson, P Monk, J Gray, H Thuraisingam

Answers on p 59.

A previously well 10 year old boy was admitted from school with increasing epigastric pain and vomiting. He had a six day history of feeling generally unwell and feverish with loose motions. On examination he was well, pyrexial at 37.6°C and mildly icteric with epigastric tenderness. There was no organomegaly. Investigations included normal full blood count, differential white count, urea, electrolytes, and chest radiography. Liver function was mildly deranged with alanine aminotransferase (ALT) 250 U/l, alkaline phosphatase (ALP) 280 U/l, and bilirubin 67µmol/l.

Paul-Bunnell test and hepatitis B surface antigen were negative. A diagnostic test was performed. He improved after 48 hours and was discharged home well.

Ten days later, a teacher from the same school presented to her general practitioner with nausea, vomiting, abdominal pain, and jaundice. On examination she was deeply icteric with no stigmata of chronic liver disease. Investigations revealed a normal full blood count with deranged liver function of ALT 1900 U/l, ALP 180 U/l, and bilirubin 142 µmol/l. Hepatitis B surface antigen and hepatitis C antibody were negative. A diagnostic test was performed.

QUESTIONS

- (1) What is the likely and differential diagnosis?
- (2) What was the diagnostic test performed?
- (3) What are the risk factors for contracting this condition?
- (4) What is the management approach to the control of this condition?

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